Phytophthora cinnamomi in Western Australia

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Phytophthora cinnamomi the agent of eucalypt dieback disease in Western Australia is a serious pathogen of many plant species around the world. The pathogen has a very wide host range. In Western Australia many species of native plants are susceptible, and a large number because they are of limited distribution are threatened with extinction. This paper reviews the mechanisms by which *P. cinnamomi* causes disease, together with the factors that contribute to the spread and survival of the pathogen allowing it cause new disease epidemics when the right conditions prevail. It also looks at possibilities for control of the pathogen by management, chemical application and biological control.

KEYWORDS: diagnostics, dieback, hemibiotroph, phosphite, *Phytophthora*.

INTRODUCTION

The genus *Phytophthora* can justifiably lay claim to containing some of the most devastating plant pathogenic species ever seen. Nearly 200 years after our first encounter with *Phytophthora* as the causative agent of the Irish Potato Famine, we have identified over 121 species in this genus (Scott *et al.* 2013) although it has been estimated that there may be as many as 500 species (Brasier 2008). These are pathogens on a very wide range of horticultural, ornamental and silvicultural species and cause major problems wherever these crops are grown. More recently we have begun to appreciate their devastating impact on native ecosystems where they infect and kill a very wide range of native species. One of the most devastating species in an Australian context is *Phytophthora cinnamomi*, the cinnamon fungus, so named as it was first identified as a pathogen of cinnamon in Sumatra by Rands (Cahill *et al.* 2008).

Although deaths of jarrah (*Eucalyptus marginata*) trees due to a dieback disease had been recorded in Western Australia since the 1920s, it was not until the 1960s that infection by *P. cinnamomi* was linked to these deaths (Podger *et al.* 1965). Subsequently *P. cinnamomi* was found to be the cause of dieback disease in forests of East Gippsland, the Brisbane Ranges, Wilsons Promontory and the Grampians in Victoria (Weste 1994). Since then 2284 of the 5710 described native plant species in the southwest corner of Western Australia have been shown to be susceptible to *P. cinnamomi* of which 800 are regarded as being highly susceptible (Shearer *et al.* 2004a). Since many of these are of localised distribution they are easily brought to the brink of extinction.

In Australia, *P. cinnamomi* is regarded as a threat to the existence of 10% of plant species currently listed as threatened under the Environment Protection and Biodiversity Conservation Act.

Despite being widely distributed within Australia, *P. cinnamomi* is considered to have been a relatively recent introduction to Australia (Cahill *et al.* 2008). This is based on the lack of resistance in native Australian species and lack of genetic diversity in the Australian *P. cinnamomi* population (Old *et al.* 1988; Dobrowolski *et al.* 2003). Plant pathogens often cause little damage to their hosts at their centre of origin having developed a natural balance through co-evolution with their hosts (Brasier 2008; Hansen 2008) However, problems arise when pathogens are introduced to other regions of the world where the checks and balances that normally keep the pathogen at bay are absent. Pathogens also tend to have a greater genetic diversity at their centre of origin (Fry *et al.* 1992). Analysis of a worldwide population of *P. cinnamomi* showed that the greatest degree of genetic diversity occurs in Papua New Guinea (Old *et al.* 1988; Dobrowolski *et al.* 2003).

TAXONOMY AND PHYLOGENY OF *PHYTOPHTHORA*

The Phylum Oomycota to which the genus *Phytophthora* belongs, display typical fungal characteristics such as mycelial growth and on this basis has traditionally been included in the Kingdom Mycota. This has complicated the development of disease management strategies, as oomycetes do not always respond well to strategies that are effective against fungal diseases. On the basis of biochemical, physiological and genome sequencing data the oomycetes are now grouped with the biflagellate heterokont (unequal flagellae) organisms in an assembly called the Stramenopiles (Hardham 2005; Tyler *et al.* 2006; Beakes *et al.* 2012). Stramenopiles together with the alveolate ciliates and the dinoflagellates constitute the Chromoalveolate Superkingdom (Beakes *et al.* 2012). Comparative genome analysis suggests a photosynthetic origin for the oomycetes. Plant pathogenic oomycetes such as *Phytophthora* are closely related to another group of Chromalveolate obligate parasites, the Apicomplexans that includes pathogenic species to humans such as the malarial parasite *Plasmodium falciparum*, and the pathogens *Cryptosporidium parvum* and *Toxoplasma gondii* the causes of cryptosporidiosis and toxoplasmosis, respectively, both of which are severe intestinal tract diseases of humans.

The most recent phylogenetic analysis of the genus *Phytophthora* encompasses 121 species divided into 10 well-supported clades (Scott *et al.* 2013). However, with the increased application of DNA sequencing technology to taxonomic studies, many species are being re-classified whilst new ones are being discovered all the time (Burgess *et al.* 2009; Scott *et al.* 2009). In the last decade the number of *Phytophthora* species has doubled (Brasier 2008).

GROWTH AND LIFE CYCLE OF *P. CINNAMOMI*

Phytophthora cinnamomi is considered to be a necrotrophic pathogen (Cahill *et al.* 2008). It grows vegetatively as a mycelium with hyphae that have few or no septa. It reproduces asexually by differentiation of the vegetative hyphae into sporangia which eventually burst to release numerous motile zoospores (Figure 1) (Hardham 2005). These are chemotactically attracted to plant roots and swim towards them and encyst on the surface. The encysted zoospores germinate by producing a germ tube that penetrates the host tissues to begin the infective cycle. Zoospores can be carried some considerable distances by moving water and are considered largely responsible for the downslope spread of the disease in favourable warm and moist environments (Cahill *et al.* 2008).

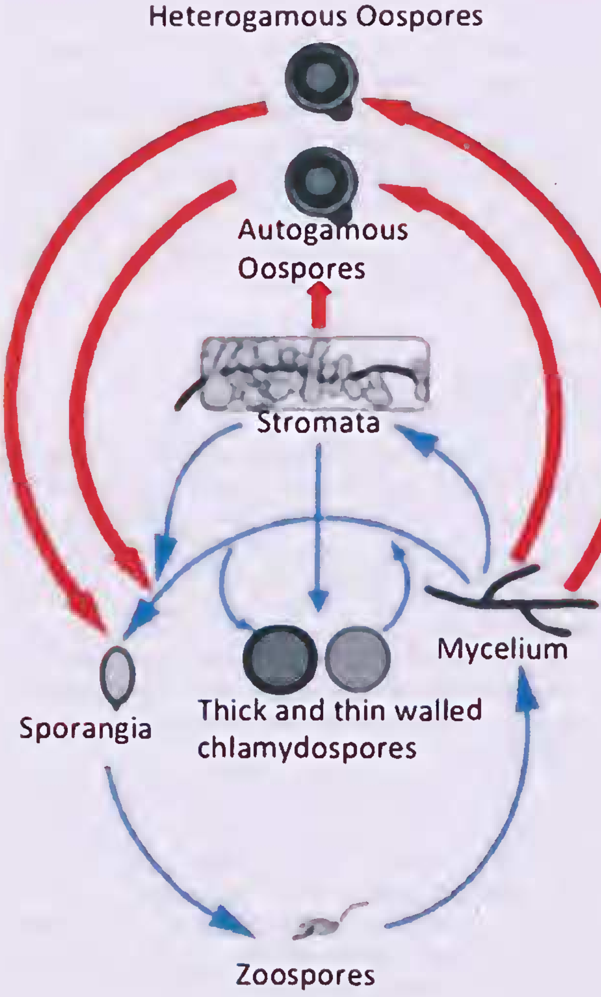


Figure 1 Life cycle of *P. cinnamomi* as modified by Crone. The life cycle is expanded from previous versions (Hardham 2005) to allow for the production of selfed oospores, stromata and thick walled chlamydospores.

The ability to reproduce asexually is probably a major factor in the success of *Phytophthora* spp. as pathogens (Shea & Broadbent 1983). In some species the sporangia can detach from the mycelium and act as a dispersal propagule for the pathogen: e.g. in *P. infestans* detached sporangia can be carried distances of several kilometres (Gregory 1983). Sporangia can also be spread by water flow or splash. Soil microorganisms stimulate sporangial production in *P. cinnamomi* (Shea & Broadbent 1983). Variations in the number of sporangial-stimulating organisms, or in the numbers of microorganisms antagonistic to them probably contribute to variation in disease development at different sites.

P. cinnamomi also produces asexual chlamydospores within the soil or planttissues. The wall of the chlamydospore is normally 0.5 μm thick, although under certain circumstances chlamydospores with especially thick walls (5 μm) are produced (Table 1) (McCarren *et al.* 2005). We have observed thick-walled chlamydospores in tissues of two herbaceous perennial species, *Chamaescilla corymbosa* and *Stylidium diuroides* and one annual *Trachymena pilosa* infected by *P. cinnamomi* in naturally infested sites in the jarrah forest in Western Australia (Crone *et al.* 2012). They have also been observed in the roots of *Banksia grandis* (Jung *et al.* 2013). *P. cinnamomi* can survive for several years in soil in the absence of a host and in the past it was generally considered that chlamydospores are the main survival structures. However, definitive evidence on this is lacking, and recent research findings indicate other survival strategies could account for the pathogen's long-term survival in infested areas.

Another type of asexual structure that occurs in *P. cinnamomi* infected tissue and has only recently been observed for the first time is the stromata (Crone *et al.* 2012; Jung *et al.* 2013). Stromata are dense intermingled hyphal aggregations that can survive adverse conditions. We have observed these to germinate *in planta* with multiple germ tubes that are capable of producing chlamydospores and selfed oospores (Crone *et al.* 2012). We speculate that stromata also serve to obtain nutrients from the host plant, which in turn allows the pathogen to produce numerous chlamydospores and selfed oospores when conditions for the pathogen become adverse. Stromata have also been observed in nine woody species found in the jarrah forest (Jung *et al.* 2013).

Sexual reproduction in *Phytophthora* occurs by fertilisation of the oogonium with a nucleus from the antheridium (Erwin & Ribeiro 1996). With heterothallic species the antheridium and oogonium are on different mycelia with different mating types (A1 and A2), whereas with homothallic species they are on the same mycelium (selfing). Although *P. cinnamomi* is considered to be heterothallic there is no genetic evidence for crossing between different mating types in natural populations despite the presence of both mating types at the same location (Dobrowolski *et al.* 2003). However, there have been several reports of the production of oospores from single isolates (selfing) of *P. cinnamomi*

Table 1 Reproductive structures observed in different asymptomatic host species infected with *Phytophthora cinnamomi.*

Host species	Reproductive structures observed							Reference
	Oospores	Chlamydospores*	Stromata	Thick-walled sclerenchyma cells	Thin-walled cells	Papillae and unbranched lignitubers	Branched lignitubers	
Eucalyptus marginata	√	a	√	√	√	√	√	Jung *et al.* 2013
Banksia grandis	√	a	√	√	√	√	√	Jung *et al.* 2013
Xanthorrhoea preissii	√	a	√	√	√	√	√	Jung *et al.* 2013
Persoonia longifolia	√	–	√	√	√	√	√	Jung *et al.* 2013
Corymbia calophylla	√	–	√	–	√	√	√	Jung *et al.* 2013
Eucalyptus megacarpa	√	–	√	–	√	√	√	Jung *et al.* 2013
Eucalyptus jaksonii	√	a	√	–	√	√	√	Jung *et al.* 2013
Acacia blakeley	√	a	√	–	√	√	√	Jung *et al.* 2013
Banksia hookeriana	√	–	–	–	√	√	√	Jung *et al.* 2013
Banksia attenuata	√	a	√	√	√	√	√	Jung *et al.* 2013
Banksia chamaephyton	–	–	√	–	√	√	√	Jung *et al.* 2013
Banksia occidentalis	√	–	–	–	√	√	–	Jung *et al.* 2013
Eremaea panciflora	√	–	–	–	√	√	√	Jung *et al.* 2013
Hakea eneabba	√	–	√	–	√	√	√	Jung *et al.* 2013
Kunzea acuminata	–	–	√	√	√	√	√	Jung *et al.* 2013
Lambertia multiflora	√	a	√	√	√	√	√	Jung *et al.* 2013
Chamaesilla corymbosa	√	b	√	–	–	–	–	Crone *et al.* 2012
Trachymena pilosa	√	b	√	–	–	–	–	Crone *et al.* 2012
Stylidium diuroides	√	b	√	–	–	–	–	Crone *et al.* 2012

* a, thick-walled chlamydospores; b, thin-walled chlamydospores

(Mircetich & Zentmeyer 1966; Jayasekera *et al.* 2007), while Crone *et al.* (2012) reported prolific oospore production in tissues of the herbaceous perennial species *Chamaecsilla corymbosa* and *Stylidium diuroides,* as well as in the annual *Trachymena pilosa.* Jung *et al.* (2013) also observed selfing to occur in a number of native Australian woody species and Mbaka *et al.* (2010) reported selfing in macadamia plants in Kenya. The selfed oospores observed in many of the native species in Western Australia had thicker walls than those that formed in mating tests between A1 and A2 types in agar culture and this may contribute to the survival of the pathogen over the hot dry Mediterranean summers in Western Australia.

The formation of selfed oospores appears to be stimulated by the presence of antagonistic organisms or by root exudates of certain species. On agar plates the presence of *Trichoderma* species stimulates selfing in *P. cinnamomi* (Brasier 1975). This effect appears to be associated with inhibition of hyphal growth by *Trichoderma.* Selfing can also be stimulated by root exudates from plants. Oleic acid present in root extracts of avocado initiated oospores in the A2 mating types of *P. cinnamomi, P. cryptogea* and *P. capsici,* but was not effective on the A1 type of *P. cinnamomi* (Zentmyer 1979). Exudates from *Acacia pulchella* stimulated oospore formation by *P. cinnamomi* in infected *Lupinus angustifolius* roots incubated for seven days under potted *Acacia pulchella* plants, or in soils collected from under and near varieties of *A. pulchella* in the jarrah forest (Jayasekera *et al.* 2007).

INTERACTION OF *P. CINNAMOMI* WITH THE HOST PLANT

The interaction with the host begins by infection of the root tips by zoospores that are chemotactically attracted to the roots. Infections can also be initiated by growth of hyphae between the roots of different species as the roots are often in contact (Shearer & Tippett 1989). Lesions may extend up to the collar, and may girdle the tree. Water transport is inhibited in susceptible but not in resistant species even though only a small proportion of the root system may be infected (Weste 1994). Once the pathogen has entered the plant tissue it rapidly colonises the wood (Davison *et al.* 1994). Colonisation of woody tissue was more extensive in pine compared to *E. marginata* (jarrah) which is relatively resistant to infection. Shearer & Tippett (1989) speculated that bark thickness may be a component of resistance although some banksias and eucalypts with thick bark are still quite susceptible (G E St J Hardy unpubl. data).

The extent of colonisation is markedly affected by environmental conditions such as temperature, moisture and the physiological status of the host. The optimal temperatures for growth in secondary phloem of *Banksia grandis* is 25–30 °C (Shearer & Tippett 1989). The length of lesions in *Banksia* correlated well with lengths predicted using previously determined growth temperature relationships. This is not the case in jarrah where growth is determined by water stress in addition to temperature. Tippett *et al.* (1987) found that trees that were well watered were more susceptible than those

suffering water stress. This was ascribed to the increased water content favouring growth of the pathogen *in planta*.

As indicated earlier, *P. cinnamomi* is considered to be a necrotrophic pathogen. Necrotrophic pathogens typically cause maceration of host tissue by bursting the cells releasing the intracellular contents on which they feed saprophytically. However, there have been a number of reports that *P. cinnamomi* does not always cause such symptoms. In a number of cases it can exist within the plant without the plant showing any adverse signs of infection. It is considered that under such conditions *P. cinnamomi* grows as a biotroph rather than as a necrotroph. This is not unusual as there are a number of examples of hemobiotriophic fungal pathogens that exhibit both types of lifestyles and can change from one to the other (Horbach *et al.* 2011). In the biotrophic mode *P. cinnamomi* appears to produce haustoria, which it uses to gain nutrients from the host (Crone *et al.* 2013).

In some cases after a hypha has penetrated the host cell wall, it may remain enveloped by a host-derived membrane, which appears as an invagination from the plasma membrane, and callose sheaths may be apposited onto this wall to encapsulate the invading hypha within the host cell (Jung *et al.* 2013). These callose sheaths called lignitubers vary in size and shape from simple spherical structures to complex branched lignitubers. The callose appositions may continue until the intracellular structure is completely encased. Callose layers are almost impermeable and, due to the incorporation of suberin, lignin and polyphenols, are highly resistant to enzymatic maceration. The elongated branched complex lignitubers have been observed in the cortex cells of the fine roots and root debris of both resistant and susceptible species. They have been found in 83% of root and root debris samples from *E. marginata* (resistant) and *B. grandis* (susceptible) in dieback sites (Jung *et al.* 2013). In samples from dieback-free sites short spherical lignitubers were observed occasionally, whilst no complex branched lignitubers were found. The involvement of lignitubers in the survival of *P. cinnamomi* is an open question. Complex lignitubers have been observed in root tissues of both *E. marginata* and *B. grandis* from which *P. cinnamomi* has been isolated after 6, 12, or 18 months dry air storage, and a lignituber from one *B. grandis* root sample has been observed to germinate giving rise to a chlamydospore (Jung *et al.* 2013).

IMPACT OF *PHYTOPHTHORA* ON THE ECOSYSTEM

Infestation of a site by *P. cinnamomi* leads to progressive death of susceptible species. However, these can take some time to die. Common flora in disease centres in the Esperance Plains Bioregion of Western Australia reached 50% mortality in five years (Shearer *et al.* 2007). This compares with more than six years for the two most common susceptible species on the Swan Coastal Plain. Eventually the susceptible species will be lost from the site. Since many of these are structurally dominant in the communities in which they occur, their removal has a dramatic effect on the community. Despite the removal of these species there is still sufficient pathogen inoculum present in symptomless or tolerant hosts to kill any regenerating susceptible species, and thus the vegetation undergoes long-term changes in favour of less-susceptible species (Cahill *et al.* 2008).

A more immediate effect of infestation by *P. cinnamomi* is a reduction in canopy cover. Shearer *et al.* (2007) reported a 27–30% reduction in canopy cover in infested forest and woodland biomes in Western Australia compared to adjacent healthy vegetation. The reduction in canopy cover can lead to overexposure of understory species with adverse consequences. An example of this is the significant reduction in ground cover of the fern *Lindsaea linearis* in an infested *Banksia* woodland compared to adjacent healthy woodland (Shearer *et al.* 2004a). As the fern is resistant to infection by *P. cinnamomi*, the effect was ascribed to increased exposure as a result of decreased canopy cover. Other plant species adversely affected by reduced canopy cover are *Stylidium scandens* in the Stirling Ranges National Park in Western Australia (Wills 1993), and several other *P. cinnamomi* resistant understory species in the jarrah forest (McDougall 2005).

Long-term monitoring of infested sites in Victoria has shown that at about 3–5 years post infestation field-resistant sedges, rushes, grasses and volunteer weeds may colonise the infested site (Weste & Marks 1987). After about 15 years the original species have disappeared. After 22 years susceptible species begin to reappear (Dawson *et al.* 1985). In Western Australia, species richness decreased in old infested areas of the jarrah forest (Shearer *et al.* 2007). Similar changes were noted in infested *Banksia* woodland (Shearer & Hill 1989), but not in infested shrubland (Shearer *et al.* 2007). The reappearance and survival of susceptible species may be determined by the persistence of the pathogen at the site. It may persist for some considerable time, and under favourable conditions may erupt and infect recently arrived susceptible species leading to a new round of vegetation changes. In Victoria the pathogen could be recovered from infested sites 15 years after infestation but not after 20–30 years (Auesukaree *et al.* 2003). In contrast, in Western Australia *P. cinnamomi* could be isolated from infested sites in the jarrah forest 50 years after the initial infestation (McDougall *et al.* 2002). This survival is now recognised to be due to the ability of *P. cinnamomi* to colonise asymptomatic and symptomatic herbaceous perennials and annuals (Crone *et al.* 2012). Consequently, the pathogen is likely able to survive indefinitely in the absence of susceptible species.

The susceptibility of a species is not uniform but varies from site to site. Thus species such as *Eucalyptus smithii*, *E. fastigata* and *E. fraxinoides* are regarded as highly susceptible to *P. cinnamomi* in commercial plantations in South Africa yet none of these species is regarded as susceptible in native habitats (Cahill *et al.* 2008). Similarly *Hibbertia hypericoides* is highly susceptible to *P. cinnamomi* on the Swan Coastal Plain of Western Australia but of low susceptibility in the jarrah forest (Shearer & Dillon 1996). The basis of this site effect is not clear but may be related to the presence of *P. cinnamomi* suppressive microorganisms at some sites (Malacjzuk 1979), or to differences in the chemical nature of the soil. In Western Australia there is a negative correlation between soil calcium content and disease incidence (Stasikowski 2012). The presence of other plant species may also affect

susceptibility: for example D'Souza *et al.* (2004) reported that *Acacia* species can protect adjacent plants of susceptible species such as *Banksia grandis* from infection. Resistance may also have a genetic component. Some native species show variation in susceptibility to *P. cinnamomi* with some varieties being quite resistant whereas others in the same species are more sensitive (Shearer *et al.* 2004a). This has also been described for a number of acacias, eucalypts, grasses, sedges, rushes and cereals (Weste & Marks 1987).

Changes in vegetation caused by *Phytophthora* dieback inevitably have an effect on the fauna that use the plants for cover, food or for nesting sites (Garkaklis *et al.* 2004). Comparative studies have shown lower numbers of invertebrates in infested sites (Nichols & Bamford 1985; Postle *et al.* 1986). Infested forest sites also supported fewer reptile and frog species compared to healthy forest sites (Nichols & Bamford 1985). Changes in vegetation also affect small mammal communities. Studies in the Brisbane Ranges in Victoria showed the abundance of *Antechinus stuartii* to be reduced in infested areas (Newell 1997). Studies in the coastal heathland at Anglesea in Victoria found that several small mammal species were also less abundant in infested coastal heathlands (S Laidlaw & B Wilson unpubl. data). A more recent study from Western Australia found that the abundance of the small marsupial, the yellow footed mardo (*Antechinus flavipes leucogaster*) was decreased in *P. cinnamomi* infested sites in the jarrah forest relative to non-infested sites (Armistead 2008). Another small marsupial whose habitat is negatively impacted by *P. cinnamomi* is the honeypossum (*Tarsipes rostratus)* (Dundas *et al.* 2013). The honeypossum feeds only on nectar and pollen and requires a high floristic diversity to maintain sustenance throughout the year. Of nine native plant species that are important in the diet of the honeypossum, five are susceptible to *P. cinnamomi.* The decreased abundance of faunal species will have ongoing effects on predatory species further up the food chain. Effectively their food sources will be diminished. In addition, as their habitat becomes more fragmented, animal populations will become more isolated and genetically less diverse. Eventually they will become too unstable.

SPREAD OF *P. CINNAMOMI*

Dispersal of *P. cinnamomi* is facilitated by moist or wet conditions and mild temperatures. The pathogen is dispersed in a variety of forms such as free zoospores in water, chlamydospores, oospores, stromata or lignitubers in soil or flowing water (Crone *et al.* 2012; Jung *et al.* 2013). It can also be dispersed in infected roots which can be moved along with the soil, or which grow from infested areas into adjacent non-infested areas (Shearer *et al.* 2004b). Therefore, any activity that causes disturbance of soil and increases water flow facilitates spread of the disease. Initially it was thought that soil disturbance was the cause of dieback because the disease occurred only at locations disturbed by activities such as mining, logging and road-building, but it was subsequently shown that such disturbance led to movement of infested soil and water and spread of the pathogen (Weste 1994). Prior to the demonstration that *P. cinnamomi* is responsible for the death of plants, gravel for roadbuilding was often unwittingly taken from infested to non-infested areas (Shearer & Tippett 1989). Propagules of *P. cinnamomi* can survive for up to 10 months in soil and gravel (Weste & Vithanage 1979). In the post-WWII period in Western Australia the increased frequency of road-building and mining activities was accompanied by an increase in the death of plants due to *P. cinnamomi* (Dell *et al.* 2005).

More recently the role of animals in the spread of *P. cinnamomi* has been investigated. Feral pigs are a major threat to the ecosystems in Western Australia. These animals roam over large areas and cause massive disturbance to the soil in their search for roots (Challies 1975; Department of Environment & Heritage 2005). These animals facilitate the spread of the disease in a number of ways: (i) they churn up huge volumes of soil bringing infested roots to the surface from where they can be more easily spread by other animals; (ii) they carry infested soil on their coats and on their trotters, sometimes for considerable distances; and (iii) the pathogen can survive passage through the pig digestive tract (Li *et al.* 2013). *P. cinnamomi* can also survive passage through the digestive tract of other animals such as birds and termites and thus these are potential vectors for disease spread (Keast & Walsh 1979).

MANAGEMENT OF *PHYTOPHTHORA* DISEASES

The aims of disease management are to reduce the effects of the pathogen in infested areas; and prevent its spread into non-infested areas. An integrated management program will consist of the following components: (i) chemical control; (ii) biological control; (iii) resistance breeding; and (iv) sanitary measures.

Chemical control

The most widely used chemical for control of oomycete pathogens is phosphite, an analogue of phosphate. Phosphite applied as an aerial spray, or as a trunk injection follows a sink–source relationship in the plant and accumulates in the roots (Guest *et al.* 1995). It prevents further colonisation of the plant by the pathogen, although it does not kill the pathogen and therefore needs to be re-applied periodically (Shearer & Fairman 1997). Foliar sprays are less effective than injections (Hardy *et al.* 2001). The percentage survival of *Banksia baxteri* and *Lambertia inermis* two years after a low-volume mist application was increased to 68% and 78%, compared to 31% and 54% in non-treated plants, respectively (Hardy *et al.* 2001). The effectiveness of phosphite application depends on the method of application, the dose applied, the plant species, the time of year it is applied (Hardy *et al.* 2001).

Biological control

Biological control involves the application of a bacterium or fungus or mixtures of microorganisms to plants with the result that they will protect the host plant from infection by pathogens. These biocontrol agents either colonise the internal tissues of the host plant as endophytes, or inhabit the zone around the root, the rhizoshpere: in some cases they do both. They protect the plant either by directly antagonising the pathogen, or by

enhancing the host's ability to ward off the infection (Ryan *et al.* 2008). The advantages of this approach are that it is non-toxic, and protection should be offered over a prolonged period, i.e. as long as the organism persists within the host. In addition because plants acquire their endophytes from adjacent plants (horizontal acquisition), this has the potential to offer protection to plants that appear after the application of the biocontrol agent (Arnold *et al.* 2003). Biocontrol is in essence, a self-perpetuating disease control system.

Soil microorganisms are known to be antagonistic to *P. cinnamomi* (Malajczuk 1983). Soils from sites in eastern and Western Australia where disease is minimal were found to contain a greater microbial load and a greater number of antagonistic bacteria and actinomycetes compared to sites where the disease was more developed. Reduced sporulation and increased hyphal lysis were observed in these soils. Malajczuk (1983) speculated that the antagonistic effect of bacteria may be due to the production of antibiotics that are active against *Phytophthora.*

Considerable effort has been expended to look for biological control agents of *Phytophthora* diseases in different crops (Table 2). Many studies report successful control using rhizobacteria, or endophytic bacteria or fungi. The impressive levels of disease control achieved in many of these studies are encouraging for the development of effective biological control strategies for *P. cinnamomi* in native ecosystems. However, considerable challenges remain as native ecosystems with a multiplicity of species are very different from horticultural or agricultural ecosystems with a single species for a limited time. One of these concerns is the duration of the protective effect. In their study on biocontrol of *Phytophthora palmivora* the agent of black pod disease in *T. cacao* by *Trichoderma* Hanada *et al.* (2009) found that the applied biocontrol agent had limited duration on the surface of the pods.

Resistance breeding

A number of native plant species show variability in susceptibility to *P. cinnamomi* in shadehouse tests which mirrors relative susceptibilities in natural environments indicating that there is a genetic basis for resistance (Shearer *et al.* 2007). Some resistant lines of jarrah have been propagated by tissue culture and are being used for rehabilitation of diseased areas (McComb *et al.* 1994; Stukely & Crane 1994). Lines of *Pinus radiata* showing high levels of resistance to *P. cinnamomi* have been identified and are being used in the pine planting program (Butcher *et al.* 1984). However, using resistance for management of disease in natural environments is a long-term prospect as resistance sources of a great many species will need to be developed to replace those plants that are lost to disease. This strategy is complicated by the increased susceptibility of *P. cinnamomi* resistant lines to other pathogens (Shearer & Tippett 1989).

Sanitary measures

Sanitary measures involve reducing the spread of soil from infested to non-infested areas. This involves controlling access to infested areas so that infested soil is not inadvertently carried into non infested areas. One option used by the Western Australian State Government is to quarantine infested areas by closing roads and restricting access (Shearer & Tippett 1989). Where access cannot be totally restricted for commercial reasons such as logging or mining, measures such as vehicle washdown stations between infested and non-infested areas, and ensuring drainage runoff from road surfaces is diverted away from non-infested areas have been important factors in limiting the spread of the pathogen.

A critical component of sanitary measures is the ability to detect the pathogen so that infested areas can be accurately mapped and infested soil and water identified and prevented from moving into healthy areas. Traditionally methods such as baiting have been used for detection (O'Brien *et al.* 2009), although recent work with molecular methods suggests that it may give many false negative results. In their study of the distribution of *P. cinnamomi* across a disease front, Williams *et al.* (2009) reported that of 336 samples that tested positive for the presence of *P. cinnamomi* by PCR (polymerase chain reaction), only seven tested positive by baiting.

Recently Dunstan *et al.* (2010) evaluated measures to reduce the spread and to eradicate the pathogen from infected areas of Cape Riche on the south coast of Western Australia. The measures included removal of

Table 2 Biological control of *Phytophthora* diseases of different host species.

Crop species	Pathogen	Antagonist	% Disease control	Reference
Capsicum	*P. capsici*	*Serratia/Chromobacter/Lysobacter*	81%	Kim *et al.* 2008
Red pepper	*Phytophthora*	*B subtilis*	86%	Lee *et al.* 2008
Capsicum	*P. capsici*	Rhizobacteria	–	Sang *et al.* 2011
Sweet pepper	*P. capsici*	*Bacillus* spp	80%	Sid *et al.* 2003
Asparagus	*P. megasperma*	*Pseudomonas aureofaciens*	55%	Godfrey *et al.* 2000
Strawberry	*P. fragariae*	Rhizobacteria	59%	Anandhakumar & Zeller 2008
Ornamental	*P. ramorum*	*Trichoderma atroviridae*	100%	Elliott *et al.* 2009
Pepper	*P. capsici*	Rhizosphere bacteria	83%	Mei *et al.* 2010
Pepper	*P. capsici*	Rhizobacteria	63%	Rajkumar *et al.* 2005
Avocado	*P. cinnamomi*	Endophytic bacteria and fungi	89%	Hakizimana *et al.* 2012
Apple	*P. cactorum*	*Penicillium*	73%	Alexander & Stewart 2001
Pepper	*P. capsici*	Rhizosphere bacteria	97%	Yuan *et al.* 2006
Cucumber	*P. drechsleri*	Rhizobacteria	85%	Maleki *et al.* 2011

vegetation either physically or by treatment with herbicides, surface and subsurface applications of fungicide and the installation of physical barriers to prevent root-to-root spread of the pathogen. The pathogen could not be detected at the site 6–9 months after application of these measures. It is considered that while the measures used here were drastic, they would be useful for treatment of spot infections that might further develop into wider infections.

CONCLUSIONS

P. cinnamomi continues to be a devastating pathogen. However, the application of molecular techniques to the study of *P. cinnamomi* in particular and to *Phytophthora* in general has greatly increased our understanding of the biology of the pathogen and how it persists and spreads. We have also been able to develop new and highly specific molecular tools for detection of *P. cinnamomi* in soil, water and plant tissue. We have developed more effective chemical treatments based on our increased understanding of how phosphite works. Other options for control such as biological control are still in the early stages of development. What are the prospects for the future? *P. cinnamomi* will always be with us. The best we can hope for is to effectively manage the disease and contain the pathogen. Critical for this is an understanding of the pathogen's biology and it is therefore crucial that research on the biology of the pathogen continues unabated. There are looming challenges with new questions: climate change, how will this affect the pathogen and its distribution? Other challenges are increases in the population of feral animals such as pigs as these lead to increased spread of the pathogen.

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